Editorial

Psychosocial factors in the aetiology of coronary heart disease

The role of psychosocial factors in the cause of coronary heart disease (CHD) has been investigated with increasing confidence over recent years, due to a number of developments. Large scale prospective studies of psychosocial factors assessed with standardised measures and using all the rigour of modern multivariate epidemiology have begun to produce results. These have been accompanied by greater sophistication in the identification of the physiological mechanisms through which emotions, behaviour, and the social environment influence disease processes. Distinctive factors may be associated with the long term progression of CHD and with the triggering of cardiac events in vulnerable individuals. Animal studies have also come of age, with older stress paradigms being replaced by observations on more relevant phenomena such as social isolation and social disruption.[1] This brief summary describes some of the main themes that are emerging in psychosocial research, and their relevance to current clinical practice.

The work environment

Psychosocial factors include both psychological characteristics such as personality, and life experiences such as acute traumatic events and long term stressors. Stressful working conditions are important in that they may induce sustained changes in neuroendocrine and autonomic activity, increasing atherogenesis. Several studies have shown that jobs characterised by high demands coupled with low control are particularly problematic, and are associated with an increased risk of CHD.[2] Data from the Whitehall II study of British civil servants indicate that low job control predicts future CHD independently of standard cardiovascular risk factors.[3] The imbalance between effort and rewards at work has also been implicated. These associations may be mediated through neuroendocrine and sympathetic nervous system activation influencing lipid metabolism, central adiposity, haemostatic processes, and cardiac function.[4] The effects are relevant to patient care and public health. Although some aspects of work can only be altered through structural change, others are influenced by individual perceptions and priorities, which can be revised with appropriate interventions.[5] These may range from brief advice from primary care teams on issues such as time management and the balance of work with other interests, to more specialist advice involving stress management.[6]

Social isolation and social support

Social isolation and a lack of emotional support have been repeatedly identified as factors related to ill health and premature mortality.[7] Kawachi *et al* studied middle aged male health professionals, free from CHD at baseline, over a four year prospective period.[8] Men who were not married, had few friends or relatives, and were not members of community groups were at increased risk for cardiovascular death after adjusting for age, smoking, history of hypertension, diabetes, hypercholesterolaemia, body mass index, family history, alcohol intake, and physical activity. The fact that associations were independent of health practices and traditional biological risk factors suggests that psychobiological processes related to emotional support may have been involved. It is not known whether social isolation can be ameliorated through professional interventions to a sufficient extent to reduce cardiovascular risk, but awareness of these factors may facilitate targeting of services.

Personality

The impact of life stress and social factors is moderated by the personality and capacity to cope psychologically. Until a decade ago, the concept of type A coronary prone behaviour dominated thinking about psychological factors and CHD. However, type A behaviour fell into disfavour after a number of studies failed to demonstrate associations with CHD.[9] The type A construct may be too diffuse, and recent work suggests that hostility is the "toxic" component related to cardiac risk. Several prospective studies have documented increased risk in individuals with a cynically hostile attitude and propensity to hostile outbursts.[10] Hostility is associated with behavioural risks such as smoking, alcohol consumption, and dietary fat intake, so part of its influence on CHD is mediated through these behaviours.[11] Self help manuals may be of use to some patients with high levels of hostility,[12] but others would benefit from specialist support.[6]

Other psychological factors have also come to the fore. The links between depression on CHD are discussed by Creed in another editorial in this series.[13] A related concept is "vital exhaustion", a state of fatigue and lack of energy that is particularly prominent in the period leading up to a cardiac event.[14] Anxiety and the inhibition of emotional expression have also been implicated, but consensus on these factors has not been reached.

Why are psychosocial factors not recognised clinically?

Cardiologists frequently state that the psychosocial factors identified in the research literature are not apparent in clinical practice. There are probably three reasons for this. First, psychosocial factors are risks rather than inevitable causes; they vary widely in importance for different patients, and will certainly not be apparent in every case. Second, psychological characteristics such as hostility may only be elicited under appropriate provocation. They are unlikely therefore to be expressed during a typical clinical consultation. Finally, there is a tendency to search for psychosocial explanations only for patients who do not have other clear risk factors such as hypertension, diabetes or smoking. Many clinicians work from implicit models that place biological and psychosocial causes as alternatives. However, psychosocial factors may be associated with other risk factors. For example, the Whitehall II study showed that social isolation, lack of control at work, and hostility are more prominent in low social class groups where smoking, insulin resistance, and other factors are clustered.[15] These better recognised risks may disguise the role of psychological features. Brief validated instruments have been developed for many of these psychosocial

factors, and could be used by a designated member of the clinical team to screen patients. This individual would provide advice to patients, and refer more extreme cases to psychiatric or psychological services.

Acute cardiac events

The notion that psychological factors trigger cardiac events is not new, but investigations have a new lease of life with developments in cardiac imaging. Techniques such as radionuclide ventriculography have been used to demonstrate "silent" myocardial perfusion deficits and left ventricular motion abnormalities during acute emotional stress in a substantial portion of patients with CHD.[16] Abnormal peripheral vasoconstrictive responses and coronary vasoconstriction at the sites of epithelial damage may both contribute.[17] Our belief that these phenomena are clinically significant has been strengthened by Holter monitoring studies showing that the occurrence of myocardial ischaemia is greater during periods of negative emotions such as tension, sadness, and frustration, independently of physical exertion.[18] Interview studies with survivors of acute myocardial infarction suggest that episodes of anger may trigger cardiac events.[19] There are exciting prospects in combining cardiological and psychosocial expertise in identifying and modifying the mechanisms responsible for acute stress effects.[20]

Conclusions

The integration of population based studies of psychosocial adversity with investigations of the mediating biological processes will extend understanding of their relevance to CHD. Current work is opening up new avenues, including the identification of the influence of emotion and behaviour on insulin resistance, platelet function, and the vascular endothelium. The recognition that an individual's circumstances and personality interact with biological risk factors may provide new opportunities for prevention, and for more refined risk stratification.

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